



**University of  
Zurich**<sup>UZH</sup>

**Zurich Open Repository and  
Archive**

University of Zurich  
University Library  
Strickhofstrasse 39  
CH-8057 Zurich  
[www.zora.uzh.ch](http://www.zora.uzh.ch)

---

Year: 2014

---

## Use of the first-person pronoun in schizophrenia

Maatz, Anke

DOI: <https://doi.org/10.1192/bjp.205.5.409>

Posted at the Zurich Open Repository and Archive, University of Zurich

ZORA URL: <https://doi.org/10.5167/uzh-101282>

Journal Article

Published Version

Originally published at:

Maatz, Anke (2014). Use of the first-person pronoun in schizophrenia. *British Journal of Psychiatry*, 205(5):409.

DOI: <https://doi.org/10.1192/bjp.205.5.409>

## Correspondence

Edited by Kiriakos Xenitidis and  
Colin Campbell

## Contents

- Use of the first-person pronoun in schizophrenia
- Early and delayed treatment of bipolar disorder
- BDNF and proBDNF as biomarkers for bipolar disorder

## Use of the first-person pronoun in schizophrenia

In their recent publication, Fineberg *et al* examined word use in first-person accounts of schizophrenia in comparison with word use in first-person accounts of mood and anxiety disorders.<sup>1</sup> One of their hypotheses concerned the use of the first-person singular pronoun 'I'. On the basis of research showing patients with mood disorders to be particularly self-focused, as well as phenomenological reports by patients suffering from schizophrenia describing a disrupted sense of self, they predicted that 'writers with schizophrenia would use "I" less often than persons with mood disorder'. They found this hypothesis to be supported by their data.

One obvious limitation of this study, admitted by the authors, is the lack of a healthy control group. Data from two such control groups, however, are readily at hand. First, one can compare the word frequencies found in their first-person accounts with their frequency in general language, as represented in reference corpora such as the Corpus of Contemporary American English.<sup>2</sup> Second, in order to compare a text format that is as similar as possible to first-person accounts of mental illness, one can make use of articles published in the *Schizophrenia Bulletin* under the rubric 'First-person account' that are not written by sufferers of schizophrenia, but by (supposedly) healthy family and friends of someone with schizophrenia (I will refer to those as 'second-person' accounts). Such comparison, based on analyses of a corpus of the *Schizophrenia Bulletin* using CQP software,<sup>3</sup> yields results that markedly differ from Fineberg *et al*'s findings (for a general introduction to corpus linguistics, see Lüdeling & Kytö<sup>4</sup>).

Since 1979, the *Schizophrenia Bulletin* has published 98 first-person accounts and 30 second-person accounts of schizophrenia. The frequency of 'I' in the first-person accounts is 34 621.67/106 words and 20 804.18/106 words in the second-person accounts. The authors of the first-person accounts use 'I' 3.34 times more often than it is used in general American English and 1.90 times more often than it occurs in general spoken American English. Comparing first- and second-person accounts, 'I' is used 1.66 times more often by people identifying as having schizophrenia spectrum disorders than by their mentally healthy friends and family members. The log likelihood test shows this difference to be significant ( $P < 0.01$ ).

Authors identifying as having schizophrenia thus use the first-person singular pronoun more often than healthy controls. Therefore, Fineberg *et al*'s finding that authors with schizophrenia use 'I' less often than authors with mood disorders does not warrant any inferences regarding pathologies of the self in schizophrenia. To further investigate the relationship between language and self-disturbances, it would be desirable to analyse linguistic data from people undergoing an acute psychotic episode as well as to consider pronouns in their wider grammatical context rather than looking at mere word frequencies.

1 Fineberg SK, Deutsch-Link S, Ichinose M, McGuiness T, Bessette AJ, Chung CK, et al. Word use in first-person accounts of schizophrenia. *Br J Psychiatry* 2014; doi: 10.1192/bjp.bp.113.140046.

- 2 Brigham Young University. *The Corpus of Contemporary American English*. Brigham Young University (<http://corpus2.byu.edu/coca>).
- 3 Hardie A. CQPweb – combining power, flexibility and usability in a corpus analysis tool. *Int J Corpus Linguist* 2012; **17**: 380–409.
- 4 Lüdeling A, Kytö M. *Corpus Linguistics – An International Handbook*. De Gruyter, 2009.

Anke Maatz, speciality trainee in psychiatry and postdoctoral research fellow, University Hospital of Psychiatry Zurich, Switzerland. Email: [anke.maatz@puk.zh.ch](mailto:anke.maatz@puk.zh.ch)

doi: 10.1192/bjp.205.5.409

**Authors' reply:** We very much appreciate the concerns Dr Maatz raises. Indeed, we raised many of them in our discussion. Here we'll take the opportunity to elaborate on our decision-making process with regard to the analyses we reported.

As Dr Maatz and we ourselves point out, we did not include a non-psychiatric control group in our analysis. We found it difficult to identify an appropriate control for our particular corpus. Writing about illness in a journal for medical professionals is a rather particular kind of enterprise that commands specific language. We considered the caregiver and family-member accounts in the *Schizophrenia Bulletin* (which Dr Maatz called 'second-person accounts'). However, we were concerned about comparing samples with different themes (writing about oneself in the first group, writing about other people in the proposed control group). That would almost certainly change pronoun use. Furthermore, family members can sometimes present with attenuated, subclinical versions of the experiences, behaviours and deficits observed in psychotic illness.<sup>2</sup> We thought these might detract from our original objective, which was to analyse word use by people with schizophrenia compared with that by individuals with another mental illness.

We agree with Dr Maatz that this comparison between two illness groups limits the conclusions we can draw. We felt we were suitably circumspect but we are happy to rehearse the point. We are gathering new data, in which process we ask standard questions of participants (including questions that engage discussion of self, others, and impersonal topics). Furthermore we are gathering those data from participants at various illness phases (prodrome, acute psychosis, chronic illness) in order to examine the hypotheses suggested by our initial study of the *Schizophrenia Bulletin* corpus.

With respect to context analysis (how words co-occur), we agree that this is an interesting and important issue. We do not think that our word-counting approach is the final word on meaning in computational linguistics (no pun intended). We are eager to analyse larger meaning structures in our corpus using the new computational techniques Dr Maatz suggests,<sup>3</sup> among others.<sup>4</sup> We look forward to reading more about the analyses of the *Schizophrenia Bulletin* corpus she mentions in the peer-reviewed literature.

Indeed, we hope that this approach, analysing the writing and speech of patients with mental illness using computational linguistics, becomes another tool employed by those committed to understanding and treating mental illness. We are glad that Dr Maatz is interested in joining us in this venture.

- 1 Callicott JH, Egan MF, Mattay VS, Bertolino A, Bone AD, Verchinski B, et al. Abnormal fMRI response of the dorsolateral prefrontal cortex in cognitively intact siblings of patients with schizophrenia. *Am J Psychiatry* 2003; **160**: 709–19.
- 2 Hardie A. CQPweb – combining power, flexibility and usability in a corpus analysis tool. *Int J Corpus Linguistics* 2012; **17**: 380–409.
- 3 Brown C, Snodgrass T, Kemper SJ, Herman R, Covington MA. Automatic measurement of propositional idea density from part-of-speech tagging. *Behav Res Methods* 2008; **40**: 540–5.

- 4 Mota NB, Vasconcelos NA, Lemos N, Pieretti AC, Kinouchi O, Cecchi GA, et al. Speech graphs provide a quantitative measure of thought disorder in psychosis. *PLoS One* 2012; **7**: e34928.

**Philip Corlett, Sarah Fineberg**, Yale University Department of Psychiatry, Connecticut Mental Health Center, New Haven, CT, USA. Email: philip.corlett@yale.edu

doi: 10.1192/bjp.205.5.409a

## BDNF and proBDNF as biomarkers for bipolar disorder

I read with great interest the recent article by Li *et al*, describing plasma levels of brain-derived neurotrophic factor (BDNF) in patients with bipolar disorder in their first depressive episode.<sup>1</sup> A total of 203 patients with a first major depressive episode, as well as 167 healthy controls, were enrolled. After 3 years of bi-annual follow-up, 164 patients with a major depressive episode completed, and of these, 21 patients were diagnosed as having bipolar disorder and 143 patients were diagnosed as having major depressive disorder. At baseline, patients with bipolar disorder and depression showed significantly lower BDNF mRNA levels ( $P < 0.001$  and  $P = 0.02$ , respectively) and plasma BDNF levels ( $P = 0.002$  and  $P = 0.01$ , respectively) compared with healthy controls. Interestingly, plasma BDNF levels in patients with bipolar disorder were lower than those in patients with depression.

This study suggests that the model for predicting bipolar disorder during a first depressive episode is a combination of BDNF mRNA with plasma BDNF levels.<sup>1</sup> BDNF (mature BDNF) is a 13 kDa polypeptide, which is initially synthesised as a precursor protein, proBDNF, in the endoplasmic reticulum. Following cleavage of the signal peptide, proBDNF (~32 kDa) is converted to mature BDNF by extracellular proteases. It was initially thought that only secreted, mature BDNF was biologically active, and that proBDNF, localised intracellularly, served as an inactive precursor. However, accumulating evidence shows that both proBDNF and mature BDNF are active, eliciting opposing effects via the p75NTR and TrkB receptors, respectively, and that both forms play important roles in several physiological functions.<sup>2</sup>

The enzyme-linked immunosorbent assay (ELISA) kits (R&D Systems) used by Li *et al* recognise both proBDNF (precursor of BDNF) and mature BDNF, because of the limited specificity of the BDNF antibody.<sup>3</sup> Using newly available human proBDNF and mature BDNF ELISA kits, which differentiate between the BDNF forms, we have reported high levels of both proBDNF and mature BDNF in human serum.<sup>3</sup> We reported that serum levels of mature BDNF, but not proBDNF, in patients with major depressive disorder were significantly lower than those in healthy controls.<sup>4</sup> And we recently found that serum levels of mature BDNF and the ratio of mature BDNF to proBDNF in mood-stabilised patients with bipolar disorder were significantly higher than in healthy controls.<sup>4</sup> Interestingly, serum levels of proBDNF in mood-stabilised patients with bipolar disorder were significantly lower than those in healthy controls.<sup>5</sup> These findings were confirmed in two independent cohorts (Sahlgrenska set and Karolinska set in Sweden).<sup>5</sup> Considering the high levels of both proBDNF and mature BDNF in human serum, and their putative opposing functions, it would be clinically and scientifically interesting to measure the individual serum levels of proBDNF and mature BDNF in this cohort study.

- Li Z, Zhang C, Fan J, Yuan C, Huang J, Chen J, et al. Brain-derived neurotrophic factor levels and bipolar disorder in patients in their first depressive episode: 3-year prospective longitudinal study. *Br J Psychiatry* 2014; **205**: 29–35.
- Hashimoto K. Brain-derived neurotrophic factor as a biomarker for mood disorders: an historical overview and future directions. *Psychiatry Clin Neurosci* 2010; **64**: 341–57.

- Yoshida T, Ishikawa M, Iyo M, Hashimoto K. Serum levels of mature brain-derived neurotrophic factor and its precursor proBDNF in healthy subjects. *Open Clin Chem J* 2012; **5**: 7–12.
- Yoshida T, Ishikawa M, Niitsu T, Nakazato M, Watanabe H, Shiraishi T, et al. Decreased serum levels of mature brain-derived neurotrophic factor (BDNF), but not its precursor proBDNF, in patients with major depressive disorder. *PLoS One* 2012; **7**: e42676.
- Södersten K, Pålsson E, Ishima T, Funa K, Landén M, Hashimoto K, et al. Abnormality in serum levels of mature brain-derived neurotrophic factor (BDNF) and its precursor proBDNF in mood-stabilized patients with bipolar disorder: a study of two independent cohorts. *J Affect Dis* 2014; **160**: 1–9.

**Kenji Hashimoto**, Professor, Chiba University Center for Forensic Mental Health. Email: hashimoto@faculty.chiba-u.jp

doi: 10.1192/bjp.205.5.410

## Declaration of interest

K.H. is a holder of the patents 'Diagnostic and examination method for eating disorder' (US 7,754,434 B2) and 'Diagnostic agent for ischemic heart disease risk group' (US 2013/0310321A1), which pertain to the measurement of BDNF as a biomarker. In addition, He has served as a scientific consultant to Astellas and Taisho and he has received research support from Abbvie, Dainippon Sumitomo, Otsuka and Taisho.

**Authors' reply:** While we agree with Professor Hashimoto's comments regarding the predictive role of mature brain-derived neurotrophic factor (mBDNF) and its precursor, proBDNF, in bipolar disorder, several points merit further discussion.

First, we presented preliminary data describing a potential role for BDNF as a biomarker for predicting bipolar disorder in major depressive disorder, although we detected the serum BDNF level using commercial kits that do not differentiate between mBDNF and proBDNF. When we reviewed the literature regarding mBDNF and proBDNF in bipolar disorder and major depressive disorder, we noticed that lower serum levels of mBDNF and higher serum levels of proBDNF were found among patients with major depressive disorder.<sup>1,2</sup> Södersten *et al* also reported that higher serum levels of mBDNF and lower proBDNF were observed among patients with bipolar disorder.<sup>3</sup> These disparate results suggest that levels of mBDNF and proBDNF, as well as the ratio of mBDNF to proBDNF, might be sensitive enough to help differentiate bipolar disorder from major depressive disorder.

Second, our previous studies indicated that BDNF probably has some sex-specific characteristics. Tang *et al*<sup>4</sup> reported that the ratio of mBDNF to proBDNF differs in a sex-specific manner in zebra finches. These findings suggest that mBDNF and proBDNF are different in males and females and should be further investigated.

Third, the findings of one of our previous studies implied that genetic interactions between genes encoding BDNF and its receptor enhance the risk of treatment-resistant depression.<sup>5</sup> Recent studies have found that mBDNF and proBDNF elicit biological effects via interaction with their respective receptors, p75NTR and TrkB. Accordingly, we concluded that evaluations of mBDNF and proBDNF should also consider their receptors. On the whole, we appreciate Professor Hashimoto's insightful comments in directing our future work.

- Yoshida T, Ishikawa M, Niitsu T, Nakazato M, Watanabe H, Shiraishi T, et al. Decreased serum levels of mature brain-derived neurotrophic factor (BDNF), but not its precursor proBDNF, in patients with major depressive disorder. *PLoS One* 2012; **7**: e42676.
- Zhou L, Xiong J, Lim Y, Ruan Y, Huang C, Zhu Y, et al. Upregulation of blood proBDNF and its receptors in major depression. *J Affect Disord* 2013; **150**: 776–84.
- Södersten K, Pålsson E, Ishima T, Funa K, Landén M, Hashimoto K, et al. Abnormality in serum levels of mature brain-derived neurotrophic factor (BDNF)

and its precursor proBDNF in mood-stabilized patients with bipolar disorder: A study of two independent cohorts. *J Affect Disorders* 2014; **160**: 1–9.

- 4 Tang YP, Wade J. Developmental changes in BDNF protein in the song control nuclei of zebra finches. *Neuroscience* 2013; **250**: 578–87.
- 5 Li Z, Zhang Y, Wang Z, Chen J, Fan J, Guan Y, et al. The role of BDNF, NTRK2 gene and their interaction in development of treatment-resistant depression: Data from multicenter, prospective, longitudinal clinic practice. *J Psychiatr Res* 2013; **47**: 8–14.

**Chen Zhang**, MD, PhD, **Zezhi Li**, MD, PhD, **Yiru Fang**, MD, PhD, Professor, Division of Mood Disorders, Shanghai Mental Health Center, Shanghai Jiao Tong University School of Medicine, Shanghai, China. Email: yirufang@gmail.com

doi: 10.1192/bjp.205.5.410a

## Early and delayed treatment of bipolar disorder

Using Danish registry data, Kessing *et al* examined the relationship between lithium response and the timing of treatment (early v. delayed).<sup>1</sup> Early treatment was associated with an increased probability of lithium response. This is a clinically important finding, given the increasing emphasis on early intervention in bipolar disorder. The results of the Kessing *et al* study are sobering. Only few patients, particularly among those for whom treatment was delayed, responded to lithium. Several factors may have contributed to the reported results.

The study did not – and possibly could not – control for the cycle shortening that is observed after successive episodes of bipolar disorder. Although the interpretation of such cycle shortening has been debated,<sup>2</sup> it is well established that early cycles are significantly longer than those occurring later; consequently, early in the course of illness one would expect longer spontaneous remissions regardless of treatment. This effect may be partially responsible for the greater treatment response in patients receiving early intervention in the Kessing *et al* study.

Naturalistic studies typically demonstrate full response in about 30% of participants<sup>3</sup> (that is, no recurrences, or the Kessing *et al* criterion, in treatment-adherent patients), which is markedly greater than the response rate observed by Kessing *et al*. This discrepancy could be related to age at first contact. The average age of participants whom Kessing *et al* reported as having received early and late treatment was 46.7 years and 49.1 years, respectively. The natural history of bipolar disorder includes an average age at onset in the second or third decade of life. The trajectory of the illness, where mania typically develops as the last stage, delays the diagnosis of bipolar disorder. Also, there is often a substantial delay in starting treatment even following the diagnosis of bipolar disorder.<sup>4,5</sup> These reports, in conjunction with the advanced age at index presentation, and high rates of antidepressant, antipsychotic and anticonvulsant use in the Kessing *et al* study suggest that participants may have been afflicted with bipolar disorder for some time before ‘first contact’. In a sample of 450 participants, Baldessarini *et al* reported a negative relationship between treatment latency and effect of treatment on time spent ill.<sup>5</sup> If the aforementioned findings are generalisable to the Danish sample, the reduced overall treatment responses may be interpreted as a consequence of relatively advanced participant age.

Finally, Kessing *et al* analysed data collected since 1995. Is it possible that participants had received lithium during the years prior? This would further complicate the interpretations of sample responsiveness to lithium, regardless of early or late initiation. In conclusion, we suggest that the findings presented by Kessing *et al* are limited by the lack of control for inter-participant differences in the manifestation of the natural history of bipolar disorder. Such control may be difficult, or in some cases impossible, to achieve using registry-based observational data, but is nevertheless imperative to understanding the effects of early v. late treatment prophylaxis in relapsing–remitting illnesses such as bipolar disorder.

- 1 Kessing LV, Vradi E, Andersen PK. Starting lithium prophylaxis early v. late in bipolar disorder. *Br J Psychiatry* 2014; **205**: 214–20.
- 2 Oepen G, Baldessarini RJ, Salvatore P, Slater E. On the periodicity of manic-depressive insanity, by Eliot Slater (1938): translated excerpts and commentary. *J Affect Disord* 2004; **78**: 1–9.
- 3 Garnham J, Munro A, Slaney C, MacDougall M, Passmore M, Duffy A, et al. Prophylactic treatment response in bipolar disorder: Results of a naturalistic observation study. *J Affect Disord* 2007; **104**: 185–90.
- 4 Ortiz A, Bradler K, Slaney C, Garnham J, Ruzickova M, O'Donovan C, et al. An admixture analysis of the age at index episodes in bipolar disorder. *Psychiatr Res* 2011; **188**: 34–9.
- 5 Baldessarini RJ, Tondo L, Hennen J. Treatment-latency and previous episodes: relationships to pretreatment morbidity and response to maintenance treatment in bipolar I and II disorders. *Bipolar Disord* 2003; **5**: 169–79.

**Abraham Nunes**, MD, MBA, **Tomas Hajek**, MD, PhD, **Martin Alda**, MD, Department of Psychiatry, Dalhousie University, Halifax, Nova Scotia, Canada. Email: malda@dal.ca

doi: 10.1192/bjp.205.5.411

**Authors' reply:** We are confident that the relatively low response rates to lithium in our study relate to the narrow definition of lithium response, rather than to characteristics of the included patients.<sup>1</sup> Thus, we intended to characterise patients who had an excellent response to lithium monotherapy; that is, patients who were ‘cured’ from further affective episodes following a start-up period of lithium as in a prior study.<sup>2</sup> We used two robust clinical indicators to define excellent lithium response: (a) lithium prescribed in monotherapy; and (b) no need for psychiatric hospital admission. By doing this, we defined lithium response in a rather rigorous way, resulting in relatively low rates of response. We do not find that our definition of lithium response hampered the finding of the study that early treatment with lithium was associated with increased probability of excellent lithium response compared with delayed treatment, or hampered the generalisability of this finding. Although cycle acceleration occurs on average in bipolar disorder<sup>3,4</sup> the results of our study may suggest that early treatment with lithium might prevent progression of bipolar disorder.

- 1 Kessing LV, Vradi E, Andersen PK. Starting lithium prophylaxis early v. late in bipolar disorder. *Br J Psychiatry* 2014; **205**: 214–20.
- 2 Kessing LV, Hellmund G, Andersen PK. Predictors of excellent response to lithium: results from a nationwide register-based study. *Int Clin Psychopharmacol* 2011; **26**: 323–8.
- 3 Kessing LV, Hansen MG, Andersen PK. Course of illness in depressive and bipolar disorders. Naturalistic study, 1994–1999. *Br J Psychiatry* 2004; **185**: 372–7.
- 4 Kessing LV, Olsen EW, Andersen PK. Recurrence in affective disorder: analyses with frailty models. *Am J Epidemiol* 1999; **149**: 404–11.

**Lars Vedel Kessing**, Psychiatric Center Copenhagen, Department O and Faculty of Health and Medical Sciences, University of Copenhagen, **Per Kragh Andersen**, Department of Biostatistics, University of Copenhagen, Denmark. Email: Lars.Vedel.Kessing@regionh.dk

doi: 10.1192/bjp.205.5.411a

## ‘Reasonable adjustments’ for vulnerable patients

We support the views of Tuffrey-Wijne & Hollins<sup>1</sup> and their argument for the NHS to take an organisational approach to embed documentation and provision of reasonable adjustments for those with protected characteristics under the Equalities Act 2010. Lord Darzi defined quality for the NHS as comprising three dimensions: safety, effectiveness and patient experience.<sup>2</sup> The provision of reasonable adjustments is central to each of these.

Safety – Tuffrey-Wijne & Hollins rightly identify the lack of provision of reasonable adjustments as being a patient safety issue. The Confidential Inquiry into Premature Deaths of People with



Learning Disabilities (CIPOLD)<sup>3</sup> demonstrated an underlying culture in which people with intellectual disabilities were disadvantaged in accessing equitable healthcare and at risk of premature death because equality for disabled people was assumed to mean treating them the same as others. It does not. Alternative methods of making services available have to be found in order to achieve equality of outcomes. Mizen *et al*, for example, demonstrated that clinical guidelines can actually increase health inequalities for people with intellectual disabilities if reasonable adjustments are not made.<sup>4</sup> If the lack of reasonable adjustments threatens to compromise safety as, in very many cases, it does for people with intellectual disabilities, this needs to be reported and reviewed as a patient safety issue.

Effectiveness – evidence put forward by Tuffrey-Wijne *et al* suggests that ward culture, staff attitudes and staff knowledge are crucial in ensuring that hospital services are accessible to vulnerable patients.<sup>5</sup> Effective care is that which is tailored to the needs of the patient, and this must involve an understanding of the adjustments they need in order to be able to receive appropriate medical and nursing care. In our view, we should go further than Tuffrey-Wijne & Hollins' requirement for Care Quality Commission inspections in England and Wales to oversee patient-specific recording of reasonable adjustments. We also need to be confident that such adjustments are being delivered, and for evidence to be provided of adequate arrangements being in place.

Patient experience – Turner & Robinson note that it is difficult for people with intellectual disabilities and their families to influence policy and practice in healthcare systems if they are not visible within them and if involvement mechanisms such as surveys and focus groups are not accessible to them.<sup>6</sup> Both the Death by Indifference<sup>7</sup> and CIPOLD reports highlighted the lack of attention paid to the views of patients and their families, preventing them from becoming active partners in their care; the CIPOLD report additionally noted the devastating impact on future care that a poor experience of healthcare can have for some people with intellectual disabilities. The provision of reasonable adjustments needs to extend to the ways in which we garner the views of people with intellectual disabilities, communicate with them, and place them at the centre of their care.

The CIPOLD report made 18 recommendations, which included (a) clear identification of people with intellectual disabilities on the NHS central registration system and in all health care records, and (b) reasonable adjustments required by, and provided to, individuals, to be audited annually and examples of best practice shared across agencies and organisations.<sup>3</sup>

It is now 4 years since the Equalities Act 2010 came into force. Our adherence to the Act must be sharpened in the light of the health inequalities faced by people with protected characteristics, including those with intellectual disabilities, so clearly demonstrated in successive reports. We all have a responsibility, and we all have a role to play, in ensuring equal outcomes for

vulnerable people through the provision of reasonable adjustments, but strong leadership is central to making it happen.

- 1 Tuffrey-Wijne I, Hollins, S. Preventing 'deaths by indifference': identification of reasonable adjustments is key. *Br J Psychiatry* 2014; **205**: 86–7.
- 2 Department of Health. *High Quality Care for All: NHS Next Stage Review Final Report*. Department of Health, 2008.
- 3 Heslop P, Blair P, Fleming P, Hoghton M, Marriott A, Russ L. The confidential inquiry into premature deaths of people with intellectual disabilities in the UK: a population-based study. *Lancet* 2014; **383**: 889–95.
- 4 Mizen L, Macfie M, Findlay L, Cooper S, Melville C. Clinical guidelines contribute to the health inequities experienced by individuals with intellectual disabilities. *Implement Sci* 2012; **7**: 42.
- 5 Tuffrey-Wijne I, Goulding L, Giatras N, Abraham E, Gillard S, White S, et al. The barriers to and enablers of providing reasonably adjusted health services to people with intellectual disabilities in acute hospitals: evidence from a mixed-methods study. *BMJ Open* 2014; **4**: e004606.
- 6 Turner S, Robinson C. *Reasonable Adjustments for People with Learning Disabilities – Implications and Actions for Commissioners and Providers of Healthcare*. Improving Health and Lives: Learning Disabilities Observatory, 2011.
- 7 Mencap. *Death by Indifference*. Mencap, 2007.

Pauline Heslop, Reader in Intellectual Disabilities Research, Matthew Hoghton, Anna Marriott, University of Bristol. Email: pauline.heslop@bristol.ac.uk

doi: 10.1192/bjp.205.5.411b

**Authors' reply:** We welcome the detailed response from Heslop *et al* giving more evidence in support of our recommendation for the effective use of reasonable adjustments during in-patient care. They also draw attention to the need for these to be properly audited by staff who understand the Equality Act 2010, which in our view would require an extensive educational programme, as there is no evidence that current audits are much more than a box-ticking exercise.

They repeat an earlier and often made recommendation that people with intellectual disabilities should be identified on a national NHS database. NHS England has already decided to set up a national learning-disability mortality review function, which will require a national database. Regrettably, this cannot commence until data linkages have been enabled by the NHS and the Health and Social Care Information Centre and it seems unlikely that this will be achieved until next summer.<sup>1</sup> Strong advocacy is needed to ensure there are no further delays in giving priority to this work.

- 1 Hansard. HL Deb 30 July 2014 vol 755 col 1583.

Sheila Hollins, St George's University of London, Irene Tuffrey-Wijne, PhD, Kingston University and Faculty of Health, Social Care and Education, St George's University of London. Email: hollins@parliament.uk

doi: 10.1192/bjp.205.5.412

## Corrections

Aripiprazole once-monthly for treatment of schizophrenia: double-blind, randomised, non-inferiority study. *BJPsych*, **205**, 135–144. Figure 3(a), p. 141: x-axis label should be 'Days from randomisation'. The online version of this paper has been corrected post-publication, in deviation from print and in accordance with this correction.

Cost-effectiveness of injectable opioid treatment v. oral methadone for chronic heroin addiction. *BJPsych*, **203**, 341–349. In the abstract,

the second sentence of the Results should read: 'Costs overall were highest for oral methadone (mean £15 805 v. £13 410 injectable heroin and £10 945 injectable methadone;  $P = n.s.$ ) due to higher costs of criminal activity'. These data were reported correctly in the body of the paper (Table 2, p. 344). The online version of this paper has been corrected post-publication, in deviation from print and in accordance with this correction.

doi: 10.1192/bjp.205.5.412a

# BJPpsych

The British Journal of Psychiatry

## Use of the first-person pronoun in schizophrenia

Anke Maatz

*BJP* 2014, 205:409.

Access the most recent version at DOI: [10.1192/bjp.205.5.409](https://doi.org/10.1192/bjp.205.5.409)

---

### References

This article cites 1 articles, 0 of which you can access for free at:

<http://bjp.rcpsych.org/content/205/5/409.1#BIBL>

### Reprints/ permissions

To obtain reprints or permission to reproduce material from this paper, please write to [permissions@rcpsych.ac.uk](mailto:permissions@rcpsych.ac.uk)

### You can respond to this article at

<http://bjp.rcpsych.org/letters/submit/bjprcpsych;205/5/409>

### Downloaded from

<http://bjp.rcpsych.org/> on February 3, 2015  
Published by The Royal College of Psychiatrists

---